Journal of Biosciences and Medicines, 2026, 14(5), 84-106
https://www.scirp.org/journal/jbm
ISSN Online: 2327-509X
ISSN Print: 2327-5081

Bacterial Contamination and Antimicrobial Resistance Profiles on Shared Computer Devices at Gulf of Sidra University, Libya

Khalifa F. Elgadari[1*], Omar S. Alqabbasi[2], Madiha W. El-Awamie[3], Nariman A. Elsharif[3]

[1]Department of Botany, Faculty of Science, Ajdabiya University, Ajdabiya, Libya
[2]Department of Molecular Diagnostics, Faculty of Biomedical Sciences, University of Benghazi, Benghazi, Libya
[3]Department of Microbiology, Faculty of Science, University of Benghazi, Benghazi, Libya
Email: *kalifa.algdari@gmail.com

How to cite this paper: Elgadari, K.F., Alqabbasi, O.S., El-Awamie, M.W. and Elsharif, N.A. (2026) Bacterial Contamination and Antimicrobial Resistance Profiles on Shared Computer Devices at Gulf of Sidra University, Libya. *Journal of Biosciences and Medicines*, **14**, 84-106.
https://doi.org/10.4236/jbm.2026.145008

Received: April 2, 2026
Accepted: May 5, 2026
Published: May 8, 2026

Abstract

In the academic environment, shared computer devices are frequently touched and may serve as reservoirs for environmental microorganisms and potential human-associated pathogens. However, data from universities in Libya remain limited. In this cross-sectional environmental microbiology study, we investigated bacterial contamination and antimicrobial resistance patterns on computer surfaces and related accessories at Gulf of Sidra University, Bin Jawad, Libya. The sampling frame comprised 35 computer devices, from which multiple surface samples were collected, resulting in 227 cultures for microbiological analysis. Specimens were obtained using sterile cotton swabs, cultured on conventional bacteriological media, identified by routine phenotypic methods with confirmatory species assignment using an automated system, and examined for antimicrobial susceptibility by disk diffusion interpreted according to CLSI guidelines. Of the 227 cultures examined, 186 (81.9%) showed microbial growth, whereas 41 (18.1%) showed no visible growth. Among growth-positive cultures, 184 of 186 (98.9%) yielded bacterial isolates, whereas two (1.1%) showed *Candida* spp. After exclusion of one isolate lacking susceptibility data, the final antimicrobial susceptibility testing cohort comprised 183 bacterial isolates. The most frequently recovered species were *Bacillus subtilis* (29.0%), *Bacillus cereus* (16.4%), and *Staphylococcus warneri* (15.8%). Resistance was not uniformly distributed across the isolate collection. The highest resistance frequencies were observed for cefixime and ceftazidime (43.2% each), followed by azithromycin (30.6%), whereas no resistant isolates were detected for amikacin, amoxicillin-clavulanic acid, ciprofloxacin, doxycycline, or imipenem. Resistance burden differed significantly across species, with the highest burden observed in *Acinetobacter haemolyticus* and the lowest in *Bacillus cereus*.

These findings suggest that shared academic computer surfaces may harbor diverse bacterial communities with structured, species-dependent resistance profiles, supporting the significance of routine hygiene measures and local environmental surveillance in the university context.

Keywords

Shared Computer Devices, Bacterial Contamination, Antimicrobial Resistance, Environmental Surveillance, University Setting, High-Touch Surfaces, AST, Libya

1. Introduction

Inanimate surfaces frequently touched by patients, students, and staff may harbor diverse microorganisms and serve as potential reservoirs for infection. Computer keyboards and other shared peripherals are among such high-contact surfaces [1]. A recent systematic meta-analysis indicated that computer keyboards and other peripherals in healthcare settings are contaminated in 24% - 100% of samples, often with skin commensals (e.g., coagulase-negative staphylococci) and sometimes with potential pathogens such as methicillin-resistant Staphylococcus aureus (MRSA), vancomycin-resistant Enterococcus (VRE), and Enterobacteriaceae [2]. Similarly, high-touchable ICU surfaces (including keyboards) routinely harbored dry biofilms loaded with microbes that included multidrug-resistant organisms (MDRs) [2]. However, the direct association between computer-surface contamination and patient infections remained poorly quantified [3]. Additionally, it has been shown that keyboards located outside patient zones could also harbor dry-surface biofilms containing MDRs, emphasizing their role as potential fomites [4].

Computers are omnipresent in university environments and a shared interface for learning, research, and administration [5]. Keyboards and accessory mice are handled extensively by multiple users, and their structural complexity, including numerous keys, crevices, and recessed surfaces, can hinder effective cleaning and disinfection [5]. In particular, multi-user keyboards have been reported to carry a greater microbial load than devices used by a single individual [6]. In academia, where dozens of students and staff use the same equipment, keyboards and mice may serve as a repository for microbes from skin, dust, and body fluids, including nosocomial pathogens. Such equipment is not necessarily disinfected regularly, and commensal and opportunistic bacteria may be able to persist on these surfaces [6]. Studies of microbial flora on computer peripherals consistently report human-derived taxa primarily. For instance, it has been reported that mobile phones and keyboards were dominated by skin commensals (coagulase-negative *Staphylococcus* spp., *Micrococcus*) or environmental *Bacillus* spp. and Enterobacteriaceae; *S. aureus* was the most commonly detected pathogen [7]. *S. epidermidis*, *Klebsiella* spp., *S. aureus*, *E coli*, and *Pseudomonas* spp. are among the common

isolates from devices at African universities [8]. These findings are consistent with several reports of data. It has been shown that most of the device contaminants are commensal, although some studies have also isolated pathogenic bacteria, including MRSA, *C. difficile*, and VRE [1]. Contamination rates are often context-dependent, such as user behavior, cleaning practices, and sampling approaches, which vary widely. Overall, shared computer peripherals in universities are frequently contaminated with coagulase-negative staphylococci and *Bacillus*, while opportunistic enterics or staphylococci are occasionally isolated [7].

A further concern is the presence of antimicrobial resistance among these environmental isolates. Dry surface biofilms may enhance the persistence of resistant bacteria on inanimate surfaces [2], a phenomenon that has been documented in intensive care units. Surveillance studies have also shown that high-touch hospital surfaces can harbor multidrug-resistant ESKAPE pathogens. For instance, Odoyo *et al.* reported that 12.6% of hospital surfaces (maternity, surgical, etc.) were contaminated with MDR *Acinetobacter baumannii*, *Klebsiella pneumoniae*, and MRSA [9]. Similarly, almost 50% of the isolates from phones and keyboard swabbed from Ghanaian students were MDR pathogens [10].

These observations emphasize that clinically relevant AMR data can be collected from environmental sampling. Integrating antimicrobial susceptibility testing into environmental studies allows evaluation of the risk associated with pathogens carried on surfaces and supports guiding infection control. Despite extensive research elsewhere, there is limited data from Libya or similar regional universities on this concern. To address this, we performed a cross-sectional survey of 35 high-touch computer keyboards and mice at Gulf of Sidra University (Bin Jawad, Libya). Samples were processed by conventional culture; isolates were identified using the traditional and automated system, and antibiotic susceptibility was assessed by disk diffusion per CLSI criteria. This study aimed to determine the frequency and types of bacteria colonizing these academic computers and to characterize any antimicrobial resistance among the isolates. These findings provide relevant evidence for informing hygiene practices and environmental infection-prevention strategies in academic settings.

2. Materials and Methods

2.1. Study Design and Setting

This was a cross-sectional laboratory-based investigation conducted to assess bacterial contamination of computer surfaces and related accessories at Gulf of Sidra University (GSU), Bin Jawad, Libya. The sampling frame comprised 35 computer devices. The study workflow included environmental surface sampling, primary culture, purification of recovered isolates, phenotypic identification using conventional bacteriological media, confirmatory organism identification using the VITEK 2 Compact system, and antimicrobial susceptibility testing by the Kirby-Bauer disk diffusion method interpreted according to CLSI M100, 34th edition. Bacterial identification by VITEK 2 Compact was used for confirmation only and

not for susceptibility testing. This level of methodological specification is consistent with current reporting expectations for transparent and reproducible observational laboratory studies.

2.2. Sample Collection and Transport

Samples were collected from the external surfaces of computer devices and related accessories at GSU using sterile cotton swabs. A separate sterile cotton swab moistened with 0.9% normal saline was used for each sampled component; accordingly, the number of swabs collected per device varied according to the number of accessible high-contact parts. Several samples were obtained from the same device, with particular attention to high-contact surfaces, including computer keyboards, mouse surfaces, mouse buttons, and other related accessories where applicable. For flat and regular surfaces, sampling was performed over an approximately 10 × 10 cm area where feasible. For irregular components, such as keyboard keys, key margins, and mouse buttons, the accessible high-touch surface was swabbed using a consistent rolling and rotating motion to maximize surface contact and microbial recovery. Devices were sampled in their routine-use condition, without disinfection immediately before swabbing; the prior routine cleaning status was not systematically recorded. Immediately after swabbing, specimens were transferred into 0.9% normal saline and transported without delay to the laboratory for microbiological processing. The source of the samples, the sampling tool, the sampled surface type, the immediate transfer medium, and the absence of transport delay are reported explicitly to ensure procedural transparency and facilitate reproducibility.

2.3. Primary Culture, Isolation, and Isolate Recovery

Upon receipt in the laboratory, swab suspensions were inoculated onto duplicate plates of blood agar and MacConkey agar for primary culture. The inoculated plates were incubated under aerobic conditions at 37˚C for 24 hours. Following incubation, visible growth was examined, and colonies with distinct morphology were subcultured as required to obtain pure bacterial isolates for downstream identification and susceptibility testing. For isolate recovery, one representative bacterial isolate was preserved from each positive bacterial culture for downstream analysis. When visible bacterial growth was observed, a representative colony was selected from the primary culture plate and subcultured to purity for identification and antimicrobial susceptibility testing. Cultures yielding Candida spp. were recorded separately and were not included in the bacterial isolate set. Accordingly, the 227 cultures yielded 184 bacterial isolates because 41 cultures showed no visible growth, and 2 yielded Candida spp. Purified isolates were subsequently subjected to further microbiological characterization. Reporting the inoculation format, incubation atmosphere, temperature, duration, and purification procedures were consistent with good practice for reproducible microbiological methods sections.

2.4. Culture Media, Phenotypic Identification, and Confirmatory Bacterial Identification

Recovered isolates were evaluated using a combination of general-purpose, selective, differential, and enriched culture media to support isolation, purification, and preliminary phenotypic characterization. The media used during laboratory processing included nutrient agar, tryptic soy agar (TSA), blood agar, and MacConkey agar for routine cultivation and recovery; eosin methylene blue (EMB) agar and mannitol salt agar (MSA) for selective and differential characterization; and chocolate agar as an enriched medium for enhanced recovery and evaluation of fastidious growth where required. Initial bacterial identification was performed manually based on colony growth characteristics on the media and the corresponding phenotypic profile produced during routine bacteriological processing. Isolates were subsequently subjected to confirmatory identification using the VITEK 2 Compact system. The VITEK 2 Compact platform was used for species identification only and was not used for antimicrobial susceptibility testing. This stepwise approach was adopted to combine conventional phenotypic bacteriology with automated confirmatory identification to confirm taxonomic assignment of the recovered isolates.

2.5. Antimicrobial Susceptibility Testing

Antimicrobial susceptibility testing was performed by the Kirby-Bauer disk diffusion method on Mueller-Hinton agar and interpreted in accordance with the Clinical and Laboratory Standards Institute (CLSI) M100, 34th edition. Zone diameter results were interpreted using the organism- and antimicrobial-specific CLSI disk diffusion criteria available in the CLSI framework for the identified isolate. For susceptibility testing, bacterial inocula were standardized to the turbidity of a 0.5 McFarland suspension, uniformly inoculated onto the surface of Mueller-Hinton agar plates, and antimicrobial discs were applied aseptically. The inoculated plates were incubated under aerobic conditions at 35˚C ± 2˚C for 16 - 18 hours, after which inhibition zone diameters were measured and interpreted according to the CLSI breakpoints. For species-antibiotic combinations without an explicitly validated CLSI interpretive criterion, results were recorded and reviewed with caution within the limits of the applicable CLSI guidance and were not over-interpreted as organism-specific clinical resistance calls beyond those criteria. The antimicrobial panel comprised amikacin (30 µg), amoxicillin-clavulanic acid (20/10 µg), azithromycin (15 µg), cefixime (5 µg), cefoxitin (30 µg), ceftazidime (30 µg), cefuroxime (30 µg), ciprofloxacin (5 µg), doxycycline (30 µg), imipenem (10 µg), trimethoprim-sulfamethoxazole (1.25/23.75 µg), and tetracycline (30 µg). For statistical analysis, AST results were recorded as categorical susceptibility outcomes and coded as susceptible (S) or resistant (R) in the analytical dataset. No additional interpretive category was retained in the final AST-evaluable dataset, and antibiotic-specific denominators were allowed to vary where susceptibility results were unavailable for individual isolates.

2.6. Definition of the AST-Evaluable Cohort and Analytical Variables

The AST-evaluable cohort was defined as all bacterial isolates recovered from the sampled cultures for which at least one antimicrobial susceptibility result was available. Cultures showing no visible growth were excluded from AST-based analyses. In addition, any bacterial isolate lacking recorded susceptibility data was excluded from the AST-evaluable cohort. Accordingly, all organism-level and antibiotic-level analyses were restricted to bacterial isolates with available AST results. For statistical analysis, susceptibility results were entered as binary categorical outcomes and coded as susceptible (S) or resistant (R). Antibiotic-specific denominators were permitted to vary when susceptibility results were unavailable for individual isolates. A derived variable, resistance burden, was constructed for each isolate and defined as the number of tested antibiotics classified as resistant. This variable was used to quantify the overall resistance profile at the isolate level and to compare resistance burden across bacterial species. Additional derived variables were generated for specific analytical objectives. Isolates were classified by bacterial species for species-level comparisons and by Gram reaction for analyses comparing Gram-positive and Gram-negative organisms. For pairwise association analyses between antibiotic resistance outcomes, only non-redundant antibiotics with variable susceptibility patterns were retained. Antibiotic variables showing complete duplication across all tested isolates were identified and handled separately in the agreement analysis rather than being retained in the non-redundant association set. In addition, for isolate-level resistance-burden analysis, duplicated antibiotic variables were not counted as separate contributors to burden. Accordingly, resistance burden was defined using non-duplicated antibiotics only.

2.7. Statistical Analysis

All statistical analyses were performed on the AST-evaluable bacterial isolate cohort using SPSS v.27. Descriptive analyses were used to summarize culture yield, isolate composition, bacterial species distribution, and antibiotic-specific susceptibility profiles. Categorical variables were summarized as frequencies and percentages, and antibiotic-specific resistance proportions were reported with 95% binomial confidence intervals using the Wilson score method. At the isolate level, resistance burden was defined as the number of antibiotics classified as resistant for each isolate. Because resistance-burden data were considered as non-parametric count-derived outcomes, comparisons across bacterial species were performed using the Kruskal-Wallis test. When the overall test was significant, Dunn's post hoc multiple-comparison procedure was applied for pairwise species comparisons, with adjustment for multiple testing by the Benjamini-Hochberg false discovery rate method. The corresponding effect size for the overall between-species comparison was expressed as epsilon-squared. Antibiotic-specific resistance frequencies were compared across bacterial species using the chi-square test of inde-

pendence for antibiotics showing variable susceptibility outcomes. Expected cell counts were examined for each contingency table, and comparisons with incomplete assumption support were retained as exploratory analyses. The magnitude of association for species-level antibiotic comparisons was quantified using Cramér's V. Antibiotics showing no variation in susceptibility outcome across isolates were not subjected to species-level comparative testing. For comparisons between Gram-positive and Gram-negative isolates, antibiotic-specific resistance frequencies were evaluated using Fisher's exact test because of sparse cell counts and imbalance in group size. Results were reported as odds ratios (ORs) with 95% confidence intervals (CIs), and continuity correction was applied where zero cells were present. Adjustment for multiple testing across antibiotic-specific Gram-group comparisons was performed using the Benjamini-Hochberg procedure.

Agreement analysis was performed to identify antimicrobial variables showing complete duplication across isolates. For this purpose, observed agreement and Cohen's kappa coefficient were calculated for antibiotic pairs with paired non-missing values. For pairwise association analysis among non-redundant antibiotics with variable susceptibility outcomes, binary resistance patterns were evaluated using the phi coefficient, with statistical significance assessed by Fisher's exact test and corrected for multiple comparisons using the Benjamini-Hochberg false discovery rate approach. All statistical tests were two-sided, and a p-value < 0.05 was considered statistically significant unless otherwise specified after multiplicity adjustment.

3. Results

3.1. Culture Positivity, Isolate Composition, and Final AST Analytical Cohort

Of the 227 cultures examined, 186 (81.9%) showed detectable microbial growth, while 41 (18.1%) showed no visible growth. Because multiple samples were obtained from the same computer device and related accessory surfaces, the primary analytical dataset for the present study was structured at the culture level rather than the device level. Accordingly, the results presented below are reported using culture-level and isolate-level denominators. Of the growth-positive cultures, 184/186 (98.9%) produced bacterial isolates, and only 2/186 (1.1%) yielded *Candida* spp. One bacterial isolate was excluded from antimicrobial susceptibility analyses because no AST result had been performed. The AST-evaluable dataset, therefore, comprised 183 bacterial isolates, representing 183/184 (99.5%) of all bacterial isolates recovered and 183/227 (80.6%) of all cultures examined. Within this dataset, susceptibility outcomes were recorded only as susceptible (S) or resistant (R), with no intermediate or other interpretive category retained. The derivation of the AST-evaluable cohort is shown in **Figure 1**, and the corresponding culture-yield and analytical denominators are presented in **Table 1**.

Table 1. Culture yield and derivation of the final AST analytical cohort.

Category	n/N	%
Cultures with microbial growth	186/227	81.9
Cultures with no growth	41/227	18.1
Bacterial isolates among growth-positive cultures	184/186	98.9
Candida spp. among growth-positive cultures	2/186	1.1
Bacterial isolates with AST data	183/184	99.5
Final AST analytical cohort among all cultures	183/227	80.6

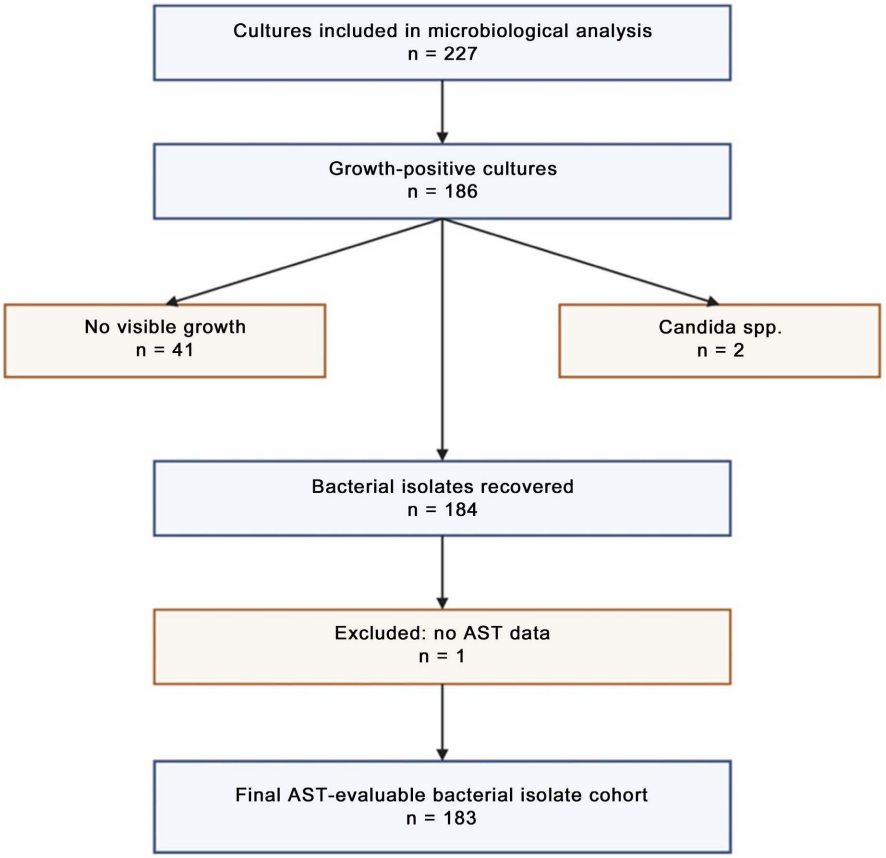

Figure 1. Derivation of the final AST-evaluable cohort from the culture dataset.

Flow diagram showing the progression from 227 retained culture observations to the final antimicrobial susceptibility testing (AST) dataset. Of the 227 cultures examined, 41 showed no growth, and 2 yielded *Candida* spp.; the remaining 184 produced bacterial isolates. After exclusion of one bacterial isolate with no AST result, the final analytic cohort comprised 183 bacterial isolates with at least one available AST result.

3.2. Bacterial Species Distribution in the Final AST Analytical Cohort

The AST-evaluable cohort comprised 183 bacterial isolates. *Bacillus subtilis* was

the most frequently recovered species, accounting for 53 isolates (29.0%), followed by *B. cereus* with 30 (16.4%) and *S. warneri* with 29 (15.8%). *S. lentus* and *Sphingomonas paucimobilis* accounted for 23 (12.6%) and 18 (9.8%) isolates, respectively. Less frequently recovered species included *S. epidermidis* (14 isolates, 7.7%), *A. haemolyticus* (10, 5.5%), and *Kocuria varians* (6, 3.3%). Together, *B. subtilis*, *B. cereus*, and *S. warneri* represented 112 of the 183 isolates (61.2%). The full species distribution is presented in Table 2.

Table 2. Distribution of bacterial species in the final AST analytical cohort (n = 183).

Bacterial Species	n	Percentage (%)
Bacillus subtilis	53	29.0
Bacillus cereus	30	16.4
Staphylococcus warneri	29	15.8
Staphylococcus lentus	23	12.6
Sphingomonas paucimobilis	18	9.8
Staphylococcus epidermidis	14	7.7
Acinetobacter haemolyticus	10	5.5
Kocuria varians	6	3.3

3.3. Overall Antimicrobial Susceptibility Profile of the Final AST Analytical Cohort

Across the 183 bacterial isolates included in the AST-evaluable cohort, susceptibility data were available for 183 isolates for most antibiotics and for 182 isolates for amikacin and imipenem. Resistance proportions were calculated using the number of isolates tested for each antibiotic, with 95% confidence intervals reported accordingly. The highest resistance frequencies were observed for cefixime and ceftazidime, each detected in 79 of 183 isolates (43.2%; 95% CI: 36.2 - 50.4). Resistance to azithromycin was identified in 56 of 183 isolates (30.6%; 95% CI: 24.4 - 37.6). Lower resistance frequencies were observed for cefuroxime, with 29 of 183 isolates resistant (15.8%; 95% CI: 11.3 - 21.8), and for tetracycline, with 14 of 183 isolates resistant (7.7%; 95% CI: 4.6 - 12.4). Resistance to cefoxitin and trimethoprim-sulfamethoxazole was uncommon and was detected in 6 of 183 isolates (3.3%; 95% CI: 1.5 - 7.0) for each agent. No resistant isolates were identified for amikacin (0/182), amoxicillin-clavulanic acid (0/183), ciprofloxacin (0/183), doxycycline (0/183), or imipenem (0/182). The antibiotic-specific susceptibility profile is presented in Table 3. The number of resistant isolates was recorded for amikacin (0/182), amoxicillin-clavulanic acid (0/183), ciprofloxacin (0/183), doxycycline (0/183), or imipenem (0/182). For these antimicrobial agents, the observed resistance proportion was 0.0%, with upper 95% confidence limits ranging to 2.1%.

Table 3. Antibiotic-specific susceptibility and resistance profile of the final AST analytical cohort.

Antibiotic	Tested Isolates (n)	Resistant, n	Susceptible, n	Resistance (%)	95% CI for Resistance (%)
Cefixime	183	79	104	43.2	36.2 - 50.4
Ceftazidime	183	79	104	43.2	36.2 - 50.4
Azithromycin	183	56	127	30.6	24.4 - 37.6
Cefuroxime	183	29	154	15.8	11.3 - 21.8
Tetracycline	183	14	169	7.7	4.6 - 12.4
Cefoxitin	183	6	177	3.3	1.5 - 7.0
Sulfamethoxazole-Trimethoprim	183	6	177	3.3	1.5 - 7.0
Amikacin	182	0	182	0.0	0.0 - 2.1
Amoxicillin-Clavulanic Acid	183	0	183	0.0	0.0 - 2.1
Ciprofloxacin	183	0	183	0.0	0.0 - 2.1
Doxycycline	183	0	183	0.0	0.0 - 2.1
Imipenem	182	0	182	0.0	0.0 - 2.1

3.4. Resistance Burden Across Bacterial Species in the Final AST Analytical Cohort

Resistance burden was defined as the number of non-duplicated antibiotics for which each isolate was classified as resistant. Across the 183 isolates in the AST-evaluable cohort, the overall median resistance burden was 1 resistant antibiotic per isolate (IQR 1 - 2; range 0 - 4). The highest burden was observed in *A. haemolyticus*, with a mean of 3.1 ± 1.2 resistant antibiotics per isolate, a median of 4, and a range of 1 - 4. *S. lentus* showed a uniform burden of 3 resistant antibiotics per isolate across all 23 isolates. *S. warneri* and *S. paucimobilis* showed a constant burden of 2 resistant antibiotics per isolate. By contrast, lower burdens were observed in *B. subtilis*, *S. epidermidis*, and *K. varians*, each with a median of 1 resistant antibiotic per isolate, whereas *B. cereus* had the lowest burden, with a median of 0 and a maximum of 1 resistant antibiotic per isolate. The distribution of resistance burden differed significantly across bacterial species (Kruskal-Wallis $H = 174.08$, $df = 7$; $p < 0.001$). Resistance burden by bacterial species is summarized in Table 4 and shown in Figure 2.

Table 4. Summary of isolate-level resistance burden by bacterial species in the final AST-evaluable cohort.

Bacterial Species	n	Mean Resistance Burden	SD	Median	IQR (Q1 - Q3)	Range
Acinetobacter haemolyticus	10	3.10	1.20	4	2 - 4	1 - 4
Staphylococcus lentus	23	3.00	0.00	3	3 - 3	3 - 3
Staphylococcus warneri	29	2.00	0.00	2	2 - 2	2 - 2
Sphingomonas paucimobilis	18	2.00	0.00	2	2 - 2	2 - 2
Bacillus subtilis	53	1.02	0.14	1	1 - 1	1 - 2

Continued

Staphylococcus epidermidis	14	1.00	0.00	1	1 - 1	1 - 1
Kocuria varians	6	1.00	0.00	1	1 - 1	1 - 1
Bacillus cereus	30	0.03	0.18	0	0 - 0	0 - 1

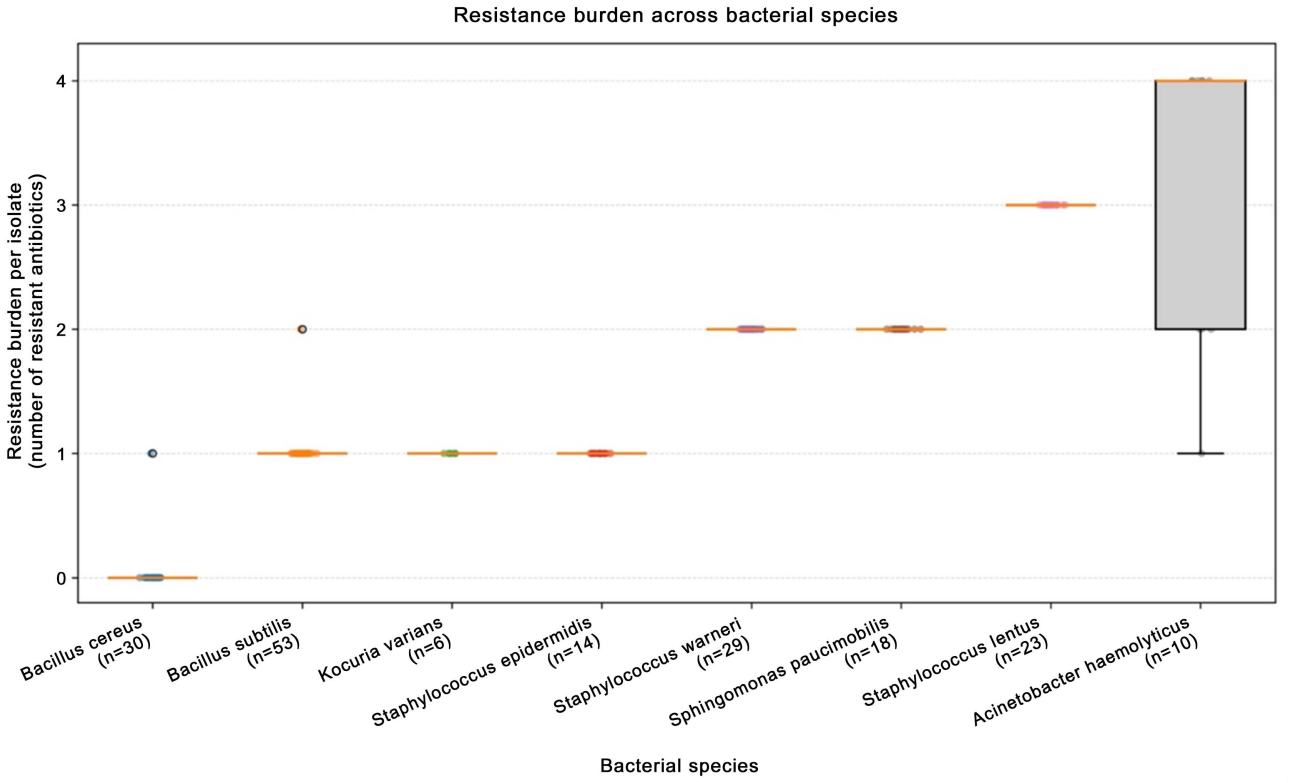

Figure 2. Distribution of resistance burden per isolate across bacterial species in the final AST analytical cohort.

Resistance burden was defined as the number of antibiotics classified as resistant for each isolate.

3.5. Species-Level Comparison of Antibiotic Resistance Frequencies

Species-level resistance patterns across the informative, non-redundant antibiotics are shown in **Figure 3**, with the corresponding comparisons presented in **Table 5**. These analyses were restricted to antibiotics that showed variation in susceptibility within the AST-evaluable cohort. For amikacin, amoxicillin-clavulanic acid, ciprofloxacin, doxycycline, and imipenem, all tested isolates were susceptible, and between-species comparisons of resistance frequency were therefore not estimable. For the remaining antibiotics, resistance frequencies differed significantly across bacterial species (all $p < 0.001$). The corresponding chi-square statistics were 143.10 for cefuroxime, 152.89 for azithromycin, 107.32 for cefoxitin, 107.32 for trimethoprim-sulfamethoxazole, 179.33 for ceftazidime, 179.33 for cefixime, and 183.00 for tetracycline, each with 7 degrees of freedom.

Table 5. Species-level comparison of antibiotic resistance frequencies in the final AST analytical cohort.

Antibiotic	Statistical Test	Test Statistic	df	p-Value	Outcome Pattern	Expected-Count Note
Doxycycline	Chi-square	—	—	—	All tested isolates were susceptible	No between-species resistance contrast was estimable
Cefuroxime	Chi-square	143.10	7	<0.001	Variable susceptibility outcome	43.8% of expected cell counts were <5; minimum expected count = 0.95
Azithromycin	Chi-square	152.89	7	<0.001	Variable susceptibility outcome	25.0% of expected cell counts were <5; minimum expected count = 1.84
Ciprofloxacin	Chi-square	—	—	—	All tested isolates were susceptible	No between-species resistance contrast was estimable
Amoxicillin-Clavulanic Acid	Chi-square	—	—	—	All tested isolates were susceptible	No between-species resistance contrast was estimable
Cefoxitin	Chi-square	107.32	7	<0.001	Variable susceptibility outcome	50.0% of expected cell counts were <5; minimum expected count = 0.20
Sulfamethoxazole-Trimethoprim	Chi-square	107.32	7	<0.001	Variable susceptibility outcome	50.0% of expected cell counts were <5; minimum expected count = 0.20
Imipenem	Chi-square	—	—	—	All tested isolates were susceptible	No between-species resistance contrast was estimable
Amikacin	Chi-square	—	—	—	All tested isolates were susceptible	No between-species resistance contrast was estimable
Ceftazidime	Chi-square	179.33	7	<0.001	Variable susceptibility outcome	18.8% of expected cell counts were <5; minimum expected count = 2.59
Cefixime	Chi-square	179.33	7	<0.001	Variable susceptibility outcome	18.8% of expected cell counts were <5; minimum expected count = 2.59
Tetracycline	Chi-square	183.00	7	<0.001	Variable susceptibility outcome	50.0% of expected cell counts were <5; minimum expected count = 0.46

Exact duplicate antibiotic columns and antibiotics with uniformly susceptible results were excluded from the display.

3.6. Agreement Analysis of Duplicated Antimicrobial Susceptibility Test Columns

Species-level agreement analysis for antimicrobial susceptibility results is presented in Table 6. Two antibiotic pairs showed complete concordance across all

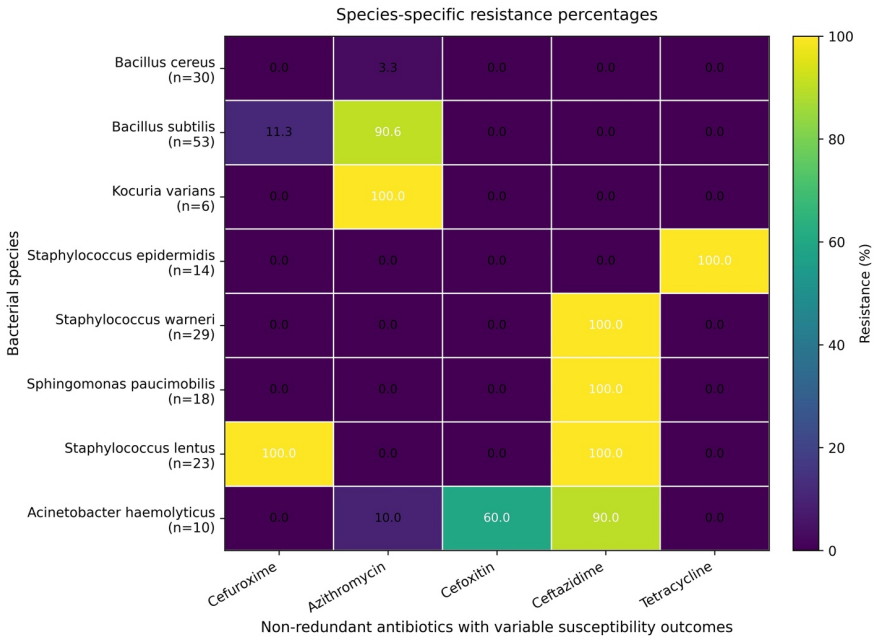

Figure 3. Heatmap of species-specific resistance percentages across non-redundant antibiotics with variable susceptibility outcomes in the final AST analytical cohort.

isolates with non-missing paired results. For cefoxitin and trimethoprim-sulfamethoxazole, agreement was assessed in 183 isolates and showed 100.0% observed agreement, with a Cohen's kappa of 1.00. An identical pattern was observed for ceftazidime and cefixime, again based on 183 paired observations, with 100.0% agreement and a Cohen's kappa of 1.00. These findings indicate complete duplication of susceptibility outcomes for both antibiotic pairs within the AST-evaluable cohort. Because these duplicated antibiotic pairs did not represent independent susceptibility variables within the dataset, they were excluded as duplicate contributors from the isolate-level resistance-burden calculation and from analyses based on non-redundant antibiotic sets.

Table 6. Agreement analysis for antimicrobial susceptibility columns showing complete duplication across tested isolates.

Column Pair	Paired Non-Missing Observations (n)	Observed Agreement (%)	Cohen's Kappa	Result
Cefoxitin vs Sulfamethoxazole-Trimethoprim	183	100.0	1.00	Identical susceptibility pattern across all tested isolates
Ceftazidime vs Cefixime	183	100.0	1.00	Identical susceptibility pattern across all tested isolates

3.7. Post Hoc Pairwise Comparison of Resistance Burden Across Bacterial Species

Following the significant overall difference in resistance burden across bacterial species, pairwise comparisons were performed using Dunn's test with Benjamini-

Hochberg correction for multiple testing. The significant adjusted comparisons are presented in Table 7. After correction, 21 of 28 pairwise comparisons remained significant. The lowest resistance burden was observed in *Bacillus cereus*, with a median burden lower than that of *Bacillus subtilis*, *Kocuria varians*, *S. epidermidis*, *S. paucimobilis*, *S. warneri*, *S. lentus*, and *A. haemolyticus*. *B. subtilis*, *K. varians*, and *S. epidermidis* also showed lower resistance burden than *S. paucimobilis*, *S. warneri*, *S. lentus*, and *A. haemolyticus* in the comparisons listed in Table 7. In addition, both *S. paucimobilis* and *S. warneri* had lower resistance burden than *S. lentus*.

Table 7. Significant Dunn post hoc pairwise comparisons of resistance burden across bacterial species after Benjamini-Hochberg adjustment.

Species with a Lower Median Burden	Species with a Higher Median Burden	Median Burden (Lower)	Median Burden (Higher)	z Statistic	Adjusted p Value
Bacillus cereus	*Bacillus subtilis*	0	1	4.42	<0.001
Bacillus cereus	*Kocuria varians*	0	1	2.21	0.036
Bacillus cereus	*Staphylococcus epidermidis*	0	1	3.05	0.004
Bacillus cereus	*Sphingomonas paucimobilis*	0	2	7.47	<0.001
Bacillus cereus	*Staphylococcus warneri*	0	2	8.55	<0.001
Bacillus cereus	*Staphylococcus lentus*	0	3	10.68	<0.001
Bacillus cereus	*Acinetobacter haemolyticus*	0	4	7.44	<0.001
Bacillus subtilis	*Sphingomonas paucimobilis*	1	2	4.46	<0.001
Bacillus subtilis	*Staphylococcus warneri*	1	2	5.26	<0.001
Kocuria varians	*Sphingomonas paucimobilis*	1	2	2.63	0.013
Kocuria varians	*Staphylococcus warneri*	1	2	2.76	0.009
Staphylococcus epidermidis	*Sphingomonas paucimobilis*	1	2	3.48	0.001
Staphylococcus epidermidis	*Staphylococcus warneri*	1	2	3.81	<0.001
Bacillus subtilis	*Staphylococcus lentus*	1	3	7.81	<0.001
Kocuria varians	*Staphylococcus lentus*	1	3	4.30	<0.001
Staphylococcus epidermidis	*Staphylococcus lentus*	1	3	5.82	<0.001
Bacillus subtilis	*Acinetobacter haemolyticus*	1	4	4.94	<0.001
Kocuria varians	*Acinetobacter haemolyticus*	1	4	3.35	0.002
Staphylococcus epidermidis	*Acinetobacter haemolyticus*	1	4	4.17	<0.001
Sphingomonas paucimobilis	*Staphylococcus lentus*	2	3	2.33	0.028
Staphylococcus warneri	*Staphylococcus lentus*	2	3	2.63	0.013

3.8. Comparison of Antibiotic Resistance Frequencies between Gram-Positive and Gram-Negative Isolates

Comparison of antibiotic resistance frequencies between Gram-positive isolates (n = 155) and Gram-negative isolates (n = 28) is presented in Table 8, and the

corresponding odds ratios with 95% confidence intervals are shown in Figure 4. Because of limited cell counts and the marked imbalance between the two groups, antibiotics with variable susceptibility outcomes were compared using Fisher's exact test. For doxycycline, ciprofloxacin, amoxicillin-clavulanic acid, imipenem, and amikacin, all tested isolates in both Gram groups were susceptible. Among antibiotics with variable outcomes, resistance frequencies differed between Gram-positive and Gram-negative isolates for cefuroxime, azithromycin, cefoxitin, trimethoprim-sulfamethoxazole, ceftazidime, and cefixime, whereas no between-group difference was identified for tetracycline. Resistance to cefuroxime was detected in 29 of 155 Gram-positive isolates (18.7%) and in none of the 28 Gram-negative isolates. Azithromycin resistance was present in 55 of 155 Gram-positive isolates (35.5%) and in 1 of 28 Gram-negative isolates (3.6%). By contrast, resistance to cefoxitin and trimethoprim-sulfamethoxazole was recorded in 6 of 28 Gram-negative isolates (21.4%), whereas no resistant Gram-positive isolate was identified for either agent. Resistance to ceftazidime and cefixime was observed in 27 of 28 Gram-negative isolates (96.4%) and in 52 of 155 Gram-positive isolates (33.5%) for each antibiotic. For tetracycline, resistance was detected in 14 of 155 Gram-positive isolates (9.0%) and in none of the 28 Gram-negative isolates.

Table 8. Comparison of antibiotic resistance frequencies between Gram-positive and Gram-negative isolates in the final AST analytical cohort.

Antibiotic	Gram-Negative Resistant/Tested n (%)	Gram-Positive Resistant/Tested n (%)	Fisher's Exact p-Value	FDR-Adjusted p-Value	Odds Ratio (95% CI)*
Doxycycline	0/28 (0.0)	0/155 (0.0)	—	—	Not estimable
Cefuroxime	0/28 (0.0)	29/155 (18.7)	0.009	0.011	0.08 (0.00 to 1.27)
Azithromycin	1/28 (3.6)	55/155 (35.5)	<0.001	<0.001	0.10 (0.02 to 0.53)
Ciprofloxacin	0/28 (0.0)	0/155 (0.0)	—	—	Not estimable
Amoxicillin-Clavulanic Acid	0/28 (0.0)	0/155 (0.0)	—	—	Not estimable
Cefoxitin	6/28 (21.4)	0/155 (0.0)	<0.001	<0.001	89.84 (4.89 to 1649.61)
Sulfamethoxazole-Trimethoprim	6/28 (21.4)	0/155 (0.0)	<0.001	<0.001	89.84 (4.89 to 1649.61)
Imipenem	0/28 (0.0)	0/154 (0.0)	—	—	Not estimable
Amikacin	0/28 (0.0)	0/154 (0.0)	—	—	Not estimable
Ceftazidime	27/28 (96.4)	52/155 (33.5)	<0.001	<0.001	36.14 (6.76 to 193.27)
Cefixime	27/28 (96.4)	52/155 (33.5)	<0.001	<0.001	36.14 (6.76 to 193.27)
Tetracycline	0/28 (0.0)	14/155 (9.0)	0.132	0.132	0.17 (0.01 to 2.95)

Points represent odds ratios, horizontal lines represent 95% confidence intervals, and the vertical reference line indicates an odds ratio of 1. Odds ratios and confidence intervals were generated using the same continuity-corrected method used for Table 8, so that both outputs reflect an identical analytical approach.

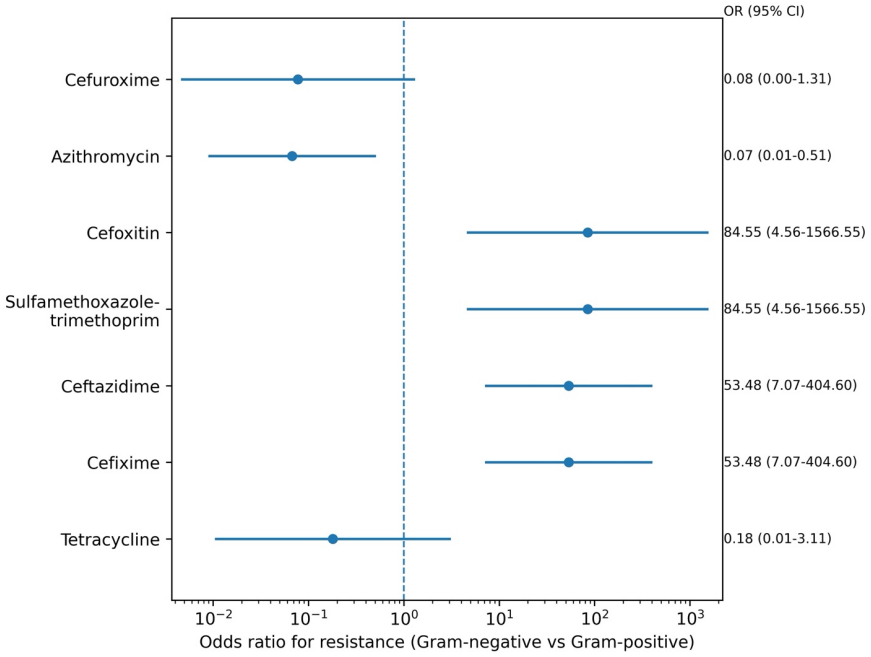

Figure 4. Odds ratios for resistance in Gram-negative relative to Gram-positive isolates across antibiotics with variable susceptibility outcomes in the final AST analytical cohort.

3.9. Pairwise Association between Resistance Outcomes among Non-Redundant Antibiotics

Pairwise associations between binary resistance outcomes are presented in **Table 9**. This analysis was restricted to non-redundant antibiotics with variable susceptibility profiles. Trimethoprim-sulfamethoxazole was excluded because its susceptibility pattern was identical to that of cefoxitin, and cefixime was excluded because it showed complete duplication with ceftazidime, as shown in **Table 6**. The association analysis, therefore, included cefuroxime, azithromycin, cefoxitin, ceftazidime, and tetracycline. Across the 10 pairwise comparisons, the strongest inverse association was observed between resistance to azithromycin and ceftazidime (phi = −0.579; FDR-adjusted $p < 0.001$). Positive associations were identified between cefuroxime and ceftazidime resistance (phi = 0.317; FDR-adjusted $p < 0.001$) and between cefoxitin and ceftazidime resistance (phi = 0.211; FDR-adjusted $p = 0.010$). Additional inverse associations were observed between cefuroxime and azithromycin (phi = −0.256; FDR-adjusted $p < 0.001$), ceftazidime and tetracycline (phi = −0.251; FDR-adjusted $p < 0.001$), and azithromycin and tetracycline (phi = −0.191; FDR-adjusted $p = 0.010$). No significant adjusted association was identified for cefuroxime versus cefoxitin, cefuroxime versus tetracycline, azithromycin versus cefoxitin, or cefoxitin versus tetracycline.

3.10. Effect Size Estimates for Between-Species Differences in Resistance Burden and Antibiotic-Specific Resistance Frequencies

Effect size estimates for the between-species comparisons are presented in **Table**

10. For overall differences in resistance burden across bacterial species, the epsilon-squared value derived from the Kruskal-Wallis test was 0.955. For antibiotic-specific comparisons of resistance frequency across species, Cramér's V was 1.000 for tetracycline, 0.990 for both ceftazidime and cefixime, 0.914 for azithromycin, 0.884 for cefuroxime, and 0.766 for both cefoxitin and trimethoprim-sulfamethoxazole. All corresponding p-values were below 0.001.

Table 9. Pairwise association between resistance outcomes among non-redundant antibiotics in the final AST analytical cohort (n = 183 for all comparisons).

Antibiotic Pair	Phi Coefficient	Fisher's Exact p-Value	FDR-Adjusted p-Value
Cefuroxime vs Azithromycin	−0.256	<0.001	<0.001
Cefuroxime vs Cefoxitin	−0.080	0.592	0.657
Cefuroxime vs Ceftazidime	0.317	<0.001	<0.001
Cefuroxime vs Tetracycline	−0.125	0.131	0.187
Azithromycin vs Cefoxitin	−0.122	0.180	0.225
Azithromycin vs Ceftazidime	−0.579	<0.001	<0.001
Azithromycin vs Tetracycline	−0.191	0.006	0.010
Cefoxitin vs Ceftazidime	0.211	0.006	0.010
Cefoxitin vs Tetracycline	−0.053	1.000	1.000
Ceftazidime vs Tetracycline	−0.251	<0.001	<0.001

Table 10. Effect size estimates for between-species comparisons in the final AST analytical cohort.

Comparison	Effect Size Metric	Effect Size Value	p-Value
Resistance burden across bacterial species	Epsilon-Squared	0.955	<0.001
Cefuroxime resistance across bacterial species	Cramér's V	0.884	<0.001
Azithromycin resistance across bacterial species	Cramér's V	0.914	<0.001
Cefoxitin resistance across bacterial species	Cramér's V	0.766	<0.001
Sulfamethoxazole-trimethoprim resistance across bacterial species	Cramér's V	0.766	<0.001
Ceftazidime resistance across bacterial species	Cramér's V	0.990	<0.001
Cefixime resistance across bacterial species	Cramér's V	0.990	<0.001
Tetracycline resistance across bacterial species	Cramér's V	1.000	<0.001

4. Discussion

In this cross-sectional study, we showed that shared computer devices and related accessories at Gulf of Sidra University were frequently contaminated with environmental and skin-associated bacteria. Across the AST-evaluable cohort, resistance patterns were clearly heterogeneous rather than uniform. Resistance was confined to a subset of antibiotics, varied substantially between bacterial species, and differed clearly between Gram-positive and Gram-negative isolates. These findings should be interpreted within the caution of an environmental surface in-

vestigation rather than evidence of clinical transmission.

The predominance of *B. subtilis* and *B. cereus* is consistent with the environment of indoor surfaces that are repeatedly exposed to dust, ambient air, and frequent hand contact. *Bacillus* species are well adapted to environmental persistence, particularly under dry conditions, and their recovery from computer keyboards and accessories is therefore biologically plausible [10] [11]. Similar patterns have been reported in studies of keyboards, mobile devices, and other frequently handled surfaces, where *Bacillus* spp. is commonly recovered alongside skin-associated staphylococci [7] [12]. In the present setting, their presence most likely reflects a combination of airborne deposition and repeated transfer from users' hands.

The predominance of coagulase-negative staphylococci is consistent with the microbial profile anticipated on high-contact academic interfaces. *S. warneri*, *S. lentus*, and *S. epidermidis* are established constituents of the resident cutaneous microbiota and therefore reflect repeated hand-mediated inoculation of shared devices. Similarly, studies of university keyboards and related peripheral equipment have likewise shown enrichment of skin-associated taxa on multi-user surfaces, particularly where routine decontamination is intermittent or suboptimal [13]-[16]. The recovery of *K. varians* supports a similar interpretation because *Kocuria* spp. are recognized members of the normal skin and mucosal flora and may also persist on environmental surfaces [17]. The less frequent recovery of *S. paucimobilis* and *A. haemolyticus* should be interpreted with caution. *S. paucimobilis* is an environmental organism associated mainly with soil and water; its isolation from shared computer surfaces is more likely to reflect environmental deposition than human carriage [18]. *A. haemolyticus* is an opportunistic pathogen mostly isolated from outside clinical settings, and therefore its presence should not be considered as evidence of healthcare-associated transmission [19]. Overall, the species profile in this study is mostly associated with environmental and commensal contamination rather than with the pathogen-dominated surface profiles described in acute-care environments [5] [6] [7] [9].

The antimicrobial susceptibility findings provided an important phenotypic complement to the surface contamination data. Resistance was selective rather than diffuse, with the main pattern confined to specific organism-antibiotic pairings. We did not observe detectable resistance to amikacin, amoxicillin-clavulanic acid, ciprofloxacin, doxycycline, or imipenem across the cohort, which is reassuring in the context of environmental surveillance, but should not be interpreted in clinical terms [17]. In contrast, resistance was more frequent for cefixime, ceftazidime, and azithromycin, and less frequent for cefuroxime, tetracycline, cefoxitin, and trimethoprim-sulfamethoxazole. This pattern suggests that resistance in the present isolates was taxonomically and phenotypically structured, rather than widespread across all recovered taxa or antimicrobial classes [8] [14].

One of the most informative findings was the marked variation in resistance burden between bacterial species. Species-level classification accounted for much

of this variation, and pairwise comparisons suggested that these differences were substantial rather than marginal. *A. haemolyticus* showed the highest burden, whereas *S. lentus* also showed a consistently elevated burden across the recovered isolates. By contrast, *B. cereus* showed the lowest burden, and *B. subtilis*, *S. epidermidis*, and *K. varians* carried comparatively limited burdens. This pattern suggests that the resistance profile detected on shared environmental surfaces is shaped less by the surface itself, but rather by the ecological traits and phenotypic characteristics of the organisms present. The consistently higher burden in *S. lentus* is particularly notable. This species has been associated with acquired resistance determinants in both clinical and environmental contexts, including macrolide resistance mechanisms [15] [16]. At the other end of the distribution, the low resistance burden observed in *B. cereus* is consistent with its recognized susceptibility profile outside specific β-lactam-associated resistance mechanisms [13] [17] [18]. Taken together, these findings suggest that resistance burden is better interpreted in relation to species composition rather than as a single aggregate measure across the entire isolate collection.

The antibiotic-specific comparisons across bacterial taxa support a similar conclusion. For all antibiotics with variable outcomes, resistance frequencies differed significantly between taxa, and the associated effect sizes were large. This suggests that resistance phenotypes were not distributed randomly across the recovered bacterial community but were clustered within specific taxa. This distinction is important for the interpretation of environmental AMR surveillance data, because surface contamination alone does not predict a uniform resistance pattern; instead, resistance is shaped predominantly by the taxonomic structure of the recovered microbiota. Although this does not change the overall interpretation, the limited expected counts reduce the accuracy of comparison between-species comparisons. Larger species-stratified datasets will be needed to refine these estimates and to assess whether the same taxonomic pattern of resistance persists over time.

The Gram-group analyses revealed similarly structured differences. Gram-positive isolates showed higher resistance frequencies for cefuroxime and azithromycin, whereas Gram-negative isolates showed higher resistance frequencies for ceftazidime, cefixime, cefoxitin, and trimethoprim-sulfamethoxazole. We did not detect a significant difference between groups for tetracycline, and several comparisons were not estimable because all isolates were susceptible. These differences are biologically plausible and consistent with known variation in intrinsic susceptibility, cell envelope structure, and taxon-specific resistance profiles. The higher azithromycin resistance in the Gram-positive fraction is consistent with the recognized importance of macrolide resistance mechanisms among staphylococci [19]. Conversely, the higher frequency of cephalosporin resistance among Gram-negative isolates is consistent with resistance profiles previously described in environmental *Sphingomonadaceae* and *Acinetobacter* spp. [20]-[23]. Pairwise analysis of resistance phenotypes provides further resolution to the dataset but does not support mechanistic inference. The inverse association between azithro-

mycin and ceftazidime resistance is consistent with the distribution of these phenotypes across different taxonomic groups in the present isolate collection. Similarly, the positive associations involving cefuroxime, cefoxitin, and ceftazidime are more likely to reflect taxon-level clustering than shared resistance mechanisms. These findings, therefore, describe co-occurrence patterns within this dataset and should not be interpreted as evidence of co-selection, genetic linkage, or horizontal gene transfer.

Complete concordance was observed between cefoxitin and trimethoprim-sulfamethoxazole, and between ceftazidime and cefixime, which reduces the independence of these variables. For the cephalosporin pair, concordant phenotypic behavior is biologically plausible given the overlapping activity profiles observed in the recovered organisms. Studies of dry-surface biofilms and persistent contamination on high-touch equipment suggest that bacterial phenotypes can remain stable on environmental surfaces, but they do not determine whether the duplicated results observed here arose from biological concordance or from the structure of the dataset [4] [8] [24]-[26].

At the institutional level, these findings are relevant, but they should be interpreted within the context of the study design. Shared computer equipment in academic environments is subject to repeated hand contact and variable cleaning, providing conditions that support the accumulation of diverse surface-associated bacterial communities. In this context, the high culture positivity observed at GSU supports the value of routine environmental hygiene measures. At the same time, the recovered flora was dominated by environmental and commensal taxa, and the study was not designed to determine transmission to users or downstream clinical risk. Its main contribution lies in showing that shared academic devices can harbor bacterial communities with structured antimicrobial resistance profiles, and that local environmental surveillance can provide evidence to inform hygiene planning and strengthen AMR awareness [8] [22].

The study has several strengths. It was based on a clearly defined sampling frame and combined culture-based recovery with isolate purification, confirmatory identification, and susceptibility interpretation according to CLSI criteria. The analytical approach also extended beyond descriptive reporting to include resistance-burden assessment, species-level and Gram-group comparisons, multiplicity adjustment, effect-size estimation, and agreement analysis. Taken together, these features strengthen the interpretive value of the dataset and provide a more informative assessment of environmental resistance patterns than prevalence estimates alone [22] [23]. There are also limitations that should be acknowledged. As a single-center cross-sectional investigation, the study provides a local snapshot rather than an established institutional pattern. We did not perform molecular analyses, and therefore, the genetic basis of the observed resistance phenotypes remains to be validated. Several subgroup comparisons were limited by sparse counts, and the duplicated AST columns reduced the independence of some antibiotic-specific inferences. The study also lacked a comparator environment and

could not distinguish transient surface deposition from persistent contamination. In addition, the inferential analyses were performed at the isolate level, and multiple isolates could have originated from the same computer device; therefore, these comparisons should be interpreted as exploratory because clustering by device was not modeled. Detailed documentation on negative swab/media controls and disk diffusion QC strain use was also not available for full reporting in the present manuscript, which limits procedural verification of the laboratory quality-control framework.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

References

[1] Bhatta, D.R., Hamal, D., Shrestha, R., Hosuru Subramanya, S., Baral, N., Singh, R.K., *et al.* (2018) Bacterial Contamination of Frequently Touched Objects in a Tertiary Care Hospital of Pokhara, Nepal: How Safe Are Our Hands? *Antimicrobial Resistance & Infection Control*, **7**, Article No. 97. https://doi.org/10.1186/s13756-018-0385-2

[2] Ide, N., Frogner, B.K., LeRouge, C.M., Vigil, P. and Thompson, M. (2019) What's on Your Keyboard? A Systematic Review of the Contamination of Peripheral Computer Devices in Healthcare Settings. *BMJ Open*, **9**, e026437. https://doi.org/10.1136/bmjopen-2018-026437

[3] Costa, D.M., Johani, K., Melo, D.S., Lopes, L.K.O., Lopes Lima, L.K.O., Tipple, A.F.V., *et al.* (2019) Biofilm Contamination of High-Touched Surfaces in Intensive Care Units: Epidemiology and Potential Impacts. *Letters in Applied Microbiology*, **68**, 269-276. https://doi.org/10.1111/lam.13127

[4] Ledwoch, K., Dancer, S.J., Otter, J.A., Kerr, K., Roposte, D. and Maillard, J. (2021) How Dirty Is Your QWERTY? The Risk of Healthcare Pathogen Transmission from Computer Keyboards. *Journal of Hospital Infection*, **112**, 31-36. https://doi.org/10.1016/j.jhin.2021.02.021

[5] Babafemi, E.O. and Church, H. (2025) The Bacterial Burden on Computer Keyboards across Selected University Facilities at Liverpool John Moores University, City Campus, Byrom Street, Liverpool. *PLOS One*, **20**, e0324977. https://doi.org/10.1371/journal.pone.0324977

[6] Anderson, G. and Palombo, E.A. (2009) Microbial Contamination of Computer Keyboards in a University Setting. *American Journal of Infection Control*, **37**, 507-509. https://doi.org/10.1016/j.ajic.2008.10.032

[7] Koscova, J., Hurnikova, Z. and Pistl, J. (2018) Degree of Bacterial Contamination of Mobile Phone and Computer Keyboard Surfaces and Efficacy of Disinfection with Chlorhexidine Digluconate and Triclosan to Its Reduction. *International Journal of Environmental Research and Public Health*, **15**, Article 2238. https://doi.org/10.3390/ijerph15102238

[8] Olu-Taiwo, M., Laryea, C.A., Kweku Mykels, D. and Forson, A.O. (2021) Multidrug-resistant Bacteria on the Mobile Phones and Computer Keyboards of Healthcare University Students in Ghana. *Canadian Journal of Infectious Diseases and Medical Microbiology*, **2021**, Article ID: 6647959. https://doi.org/10.1155/2021/6647959

[9] Odoyo, E., Matano, D., Tiria, F., Georges, M., Kyanya, C., Wahome, S., *et al.* (2023) Environmental Contamination across Multiple Hospital Departments with Multi-

drug-Resistant Bacteria Pose an Elevated Risk of Healthcare-Associated Infections in Kenyan Hospitals. *Antimicrobial Resistance & Infection Control*, **12**, Article No. 22. https://doi.org/10.1186/s13756-023-01227-x

[10] Hamiot, A., Lemy, C., Krzewinski, F., Faille, C. and Dubois, T. (2023) Sporulation Conditions Influence the Surface and Adhesion Properties of *Bacillus subtilis* Spores. *Frontiers in Microbiology*, **14**, Article 1219581. https://doi.org/10.3389/fmicb.2023.1219581

[11] Wood, J.P., Meyer, K.M., Kelly, T.J., Choi, Y.W., Rogers, J.V., Riggs, K.B., *et al.* (2015) Environmental Persistence of *Bacillus anthracis* and *Bacillus subtilis* Spores. *PLOS ONE*, **10**, e0138083. https://doi.org/10.1371/journal.pone.0138083

[12] Fukada, T., Iwakiri, H. and Ozaki, M. (2008) Anaesthetists' Role in Computer Keyboard Contamination in an Operating Room. *Journal of Hospital Infection*, **70**, 148-153. https://doi.org/10.1016/j.jhin.2008.05.023

[13] Ziogou, A., Giannakodimos, I., Giannakodimos, A., Baliou, S. and Ioannou, P. (2023) *Kocuria* Species Infections in Humans—A Narrative Review. *Microorganisms*, **11**, Article 2362. https://doi.org/10.3390/microorganisms11092362

[14] Nandy, S., Dudeja, M., Das, A.K. and Tiwari, R. (2013) Community Acquired Bacteremia by *Sphingomonas paucimobilis*: Two Rare Case Reports. *Journal of Clinical and Diagnostic Research*, **7**, 2947-2949. https://doi.org/10.7860/jcdr/2013/6459.3802

[15] Akbulut, S., Yilmaz, F. and Icgen, B. (2014) Surface Water Isolates of Hemolytic and Non-Hemolytic *Acinetobacter* with Multiple Drug and Heavy Metal Resistance Ability. *Journal of Water and Health*, **12**, 1-12. https://doi.org/10.2166/wh.2013.304

[16] Bottone, E.J. (2010) *Bacillus cereus*, a Volatile Human Pathogen. *Clinical Microbiology Reviews*, **23**, 382-398. https://doi.org/10.1128/cmr.00073-09

[17] Hart, A., Warren, J., Wilkinson, H. and Schmidt, W. (2023) Environmental Surveillance of Antimicrobial Resistance (AMR), Perspectives from a National Environmental Regulator in 2023. *Eurosurveillance*, **28**, Article ID: 2200367. https://doi.org/10.2807/1560-7917.es.2023.28.11.2200367

[18] Aarestrup, F.M. and Jensen, L.B. (2000) Presence of Variations in Ribosomal Protein L16 Corresponding to Susceptibility of Enterococci to Oligosaccharides (Avilamycin and Evernimicin). *Antimicrobial Agents and Chemotherapy*, **44**, 3425-3427. https://doi.org/10.1128/aac.44.12.3425-3427.2000

[19] Ohji, G., Oba, Y., Takimoto, K., Kagawa, H., Uchida, D., Suganaga, R., *et al.* (2010) *Bacillus cereus* Bacteremia in Hospital Setting. *International Journal of Infectious Diseases*, **14**, e388. https://doi.org/10.1016/j.ijid.2010.02.484

[20] Pleshko, E.M. and Zhurina, M.V. (2025) Distribution of Antibiotic Resistance Genes in *Kocuria* Species. *Antibiotics*, **14**, Article 1041. https://doi.org/10.3390/antibiotics14101041

[21] Miklasińska-Majdanik, M. (2021) Mechanisms of Resistance to Macrolide Antibiotics among *Staphylococcus aureus*. *Antibiotics*, **10**, Article 1406. https://doi.org/10.3390/antibiotics10111406

[22] Vaz-Moreira, I., Nunes, O.C. and Manaia, C.M. (2011) Diversity and Antibiotic Resistance Patterns of *Sphingomonadaceae* Isolates from Drinking Water. *Applied and Environmental Microbiology*, **77**, 5697-5706. https://doi.org/10.1128/aem.00579-11

[23] Müller, C., Reuter, S., Wille, J., Xanthopoulou, K., Stefanik, D., Grundmann, H., *et al.* (2023) A Global View on Carbapenem-Resistant *Acinetobacter baumannii*. *mBio*, **14**, e02260-23. https://doi.org/10.1128/mbio.02260-23

[24] Spanu, T., Sanguinetti, M., Ciccaglione, D., D'Inzeo, T., Romano, L., Leone, F., *et al.*

(2003) Use of the VITEK 2 System for Rapid Identification of Clinical Isolates of Staphylococci from Bloodstream Infections. *Journal of Clinical Microbiology*, **41**, 4259-4263. https://doi.org/10.1128/jcm.41.9.4259-4263.2003

[25] Joyanes, P., del Carmen Conejo, M., Martínez-Martínez, L. and Perea, E.J. (2001) Evaluation of the VITEK 2 System for the Identification and Susceptibility Testing of Three Species of Nonfermenting Gram-Negative Rods Frequently Isolated from Clinical Samples. *Journal of Clinical Microbiology*, **39**, 3247-3253. https://doi.org/10.1128/jcm.39.9.3247-3253.2001

[26] Centeleghe, I., Norville, P., Hughes, L. and Maillard, J. (2022) Dual Species Dry Surface Biofilms; Bacillus Species Impact on Staphylococcus Aureus Survival and Surface Disinfection. *Journal of Applied Microbiology*, **133**, 1130-1140. https://doi.org/10.1111/jam.15619